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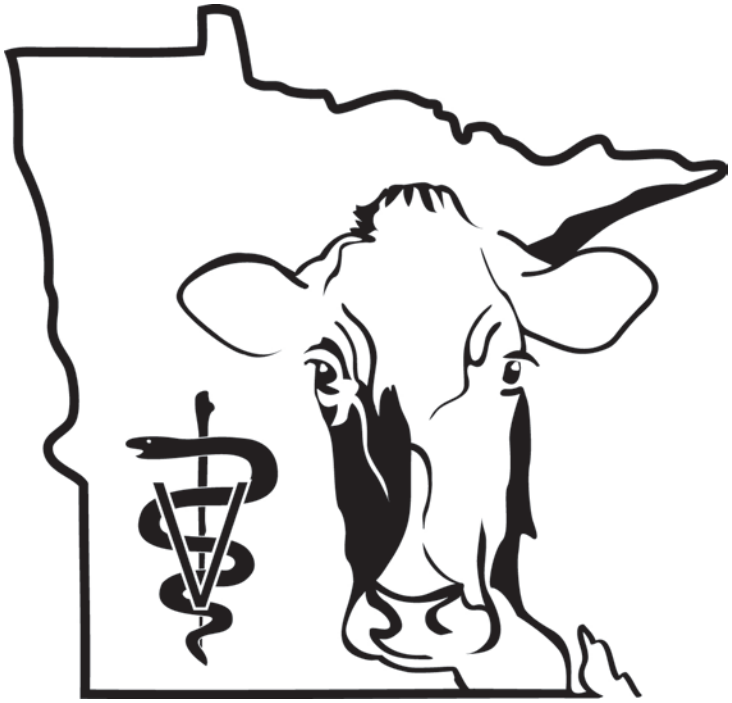

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ACUTE AND ENDEMIC BVD

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Bovine virus (viral) diarrhea (BVD) was first described following disease outbreaks in New York State and Canada during the mid 1940's. Since then veterinarians have alternated between confidence and bewilderment in their understanding of the complexity of the diseases associated with the BVD virus. One approach to improving understanding of BVD is to separate BVD virus associated diseases into two groups, those due to prenatal infections and those due to postnatal infection. (A list definitions is appended to this article)

Mucosal disease and other prenatal infections:

Mucosal disease (either chronic or acute) occurs in cattle that are first infected with BVD virus as a fetus. If a fetus becomes infected before 120 to 150 days of gestation, they may be born persistently infected with BVD virus. These calves are lifelong carriers of the virus. They may be clinically normal and inapparent carriers or they may be chronic poor-doers. Eventually, they develop acute or chronic mucosal disease and die as a result. They often developed mucosal disease (MD) by 18 to 24 months of age although some live for years before succumbing to MD.

A noncytopathic biotype of BVD virus can be consistently isolated from the plasma and leukocytes of persistently infected carriers. Both cytopathic and noncytopathic biotypes of BVD virus can be isolated from the blood or tissues of cattle with acute and chronic MD. The two biotypes of the virus isolated from cattle with acute MD can be shown to have nearly identical antigenic structures. This similarity has led to an hypothesis that the cytopathic virus arises from the noncytopathic isolate and leads to the development of clinical mucosal disease.

Persistently infected carriers are important in maintaining BVD virus in the cattle population. They continuously shed BVD virus in their body fluids. When they contact other cattle, they pass along the BVD virus. They appear to play the major role in keeping BVD virus in the cattle population by transmitting BVD both within and between herds. Their importance in some herds is emphasized by the fact that it is virtually impossible to eradicate BVD until all carriers have been identified and eliminated.

BVD virus infection of pregnant cows does not always result in the birth of carrier calves. Other outcomes include reproductive failure from early embryonic death, abortion and still birth. Calves infected in utero may also be born weak or stunted. Calves may be born with congenital defects; particularly defects of the central nervous system. The outcome of prenatal infection depends on a variety of factors including the stage of gestation, the particular isolate of the virus and the immune status of the dam.

In Ontario, extension veterinarians consult on several herds each year that have chronic problems suspected to be due to BVD. These herds have a range of clinical complaints usually going back several years. Often these herds have reproductive complaints such as poor conception rates, high

rates of repeat breeders and sporadic abortions. Frequently there are chronic health problems such as ill thrift, chronic pneumonia and diarrhea in calves. Calf health can be improved by management changes such as switching to hutches but these changes rarely completely eliminate the complaints. There is often a history of sporadic deaths from acute or chronic mucosal disease in animals under a year of age. It is unusual to have more than 1 or 2 animals die from mucosal disease each year in these herds.

A big challenge in investigating these herds is to determine if BVD is causing or contributing to the problem. Post mortem, serology and virus isolation are all beneficial in trying to confirm that the problem is due to BVD. Strategic serologic testing of unvaccinated heifers may be used (sentinel heifers). Monitoring the BVD antibody titre of several 6 to 8 month old heifers as colostral immunity wanes and before they are vaccinated can give insight into whether BVD carriers are active in the herd. If these heifers have antibody against BVD virus, it suggests that they have been exposed to the virus, most probably through contact with carriers in the herd.

Once a diagnosis of BVD is made, we advise the producer that carriers are likely present in the herd and that these carriers are maintaining the virus in the herd. These herds have usually been vaccinating to try to control BVD, but it is difficult to control BVD with vaccination alone. If vaccination does not reduce the reproduction or health complaints to an acceptable level, carriers should be identified and culled to slaughter. If the producer seriously wishes to be free of BVD and accepts that they must cull any carriers, we advise them to identify carriers by virus isolation from serum or buffy coat. Virus isolation is the most reliable method of identification. We do not recommend using serology to identify seronegative negative cattle as candidates for further testing because it is not reliable. Virus isolation is more accurate and consumes about the same resources as virus neutralization serology. Owners should commit to culling all carriers before any testing. When testing for carriers, it is essential that the samples be collected and transported promptly.

We recommend that every bovine on the farm be screened for BVD virus. Age or clinical condition are no assurance that an animal is or is not persistently infected. The herd sire or clean up bull is often missed but they should be tested too. As part of the test procedure, we recommend that all calves born on the farm should also be tested until 275 days after the last positive animal was removed. Calves should be kept isolated from pregnant cattle until they test negative. The provincial veterinary laboratories wash buffy coat samples collected using EDTA from calves to remove colostral antibodies that might interfere with the virus isolation procedure. We advise producers that there is a possibility that no carriers may be identified during herd screening.

If it is possible, we recommend retesting all cattle that were positive on virus isolation to make sure they are persistently infected carriers rather than acutely infected. If they are still virus positive 3 to 4 weeks after the first test, they are considered persistently infected. Acutely infected animals are likely to be negative on the second test. In our experience, there are usually only a few carriers on a farm. There may be a large number of carriers on one farm if many cattle are bred at the same time as occurs with embryo transfer or following estrus synchronization.

Between 1991 and 1993, 26 herds suspected of having endemic BVD were screened for carriers by virus isolation (data provided by Dr. S. Carman, Veterinary Laboratory Services, Guelph). Thirty two carriers were detected from a total of 1499 cattle tested (rate of 2.13%). Examples of carrier identification rates for some individual herds are:

- number positive/number tested
  - 0/57 (6 deaths form MD before testing)
  - 5/89 (problems resolved by removal)
  - 3/25 (abortion storm 9 months before testing)
  - 2/119 (problems resolved by removal)
  - 0/109 (no clear evidence of BVD before testing)
  - 3/90 (problem identified using sentinel heifers)
  - 0/47 (sentinel heifers seronegative)
  - 2/90 (5 deaths from MD)

In herds **without** endemic or chronic BVD problems, the general control recommendation for BVD is aimed at protecting the fetus from infection. Vaccination of all breeding age females has been recommended. Ideally, they should be vaccinated before breeding because BVD virus is a risk throughout gestation. Many producers in Ontario complain that they do not have a large enough herd to make the routine vaccination of fresh cows before breeding a practical option. It is also difficult to prove that this recommendation improves the effectiveness of the control program. Most vaccinate once yearly as a compromise but we recommend using a strategic vaccination plan to optimize immunity before breeding.

The extent that the fetus is protected by vaccinating the dam is not known. There are no label claims that vaccination will protect the fetus. Research evidence indicates that circulating antibody in the cow can protect the fetus but there are published descriptions of abortions and the birth of persistently infected calves in vaccinated herds. Certainly, there is no assurance that vaccination will be effective in protecting the fetus of an individual vaccinated animal. Vaccination increases the overall immunity of the herd. An additional unknown in recommending strategic vaccination programs to protect dairy herds is the duration of immunity. How often should vaccination be boosted to maintain protection in breeding animals? The labels recommend an annual booster.

Acute postnatal BVD virus infection:

The outcome of infection with BVD viruses in postnatal life is variable. It depends on the resistance of the host and the virulence characteristics of the virus. Most often acute infection is subclinical or results in only mild clinical signs. Acute infection can impair immune system function and render cattle more susceptible to infection by other agents. Some acutely infected animals may be more likely to develop clinical disease, develop chronic disease or respond poorly to treatment. This affect of BVD viruses can be shown experimentally. Calves co-infected with BVD and another pathogen are more severely affected than calves infected with the pathogen alone.

In some instances, acute BVD virus infection can lead to clinical disease but affected animals usually recover. Rarely is infection severe enough to cause death. A well documented example of severe disease is the syndrome of acute BVD with thrombocytopenia in veal calves. The CD87 BVD virus isolate has been used experimentally to reproduce consistently the syndrome of thrombocytopenia in calves. This virus was first isolated from a dairy herd in New York state where it cause severe clinical disease in adult cows.

Until recently, acute BVD has not been a major health concern in Ontario. In mid 1993, BVD began to be reported much more commonly than before. The number of diagnostic submissions to the provincial veterinary diagnostic laboratories helps define the change. In 1991 and 1992, the rate of diagnosis of BVD related diseases was constant at about 15 BVD diagnoses in every 500 bovine submissions. In 1993, the number of bovine diagnostic submissions has remained the same as in previous years, but the rate of BVD diagnosis has increased to 37 per 500 bovine submissions. The monthly rate began to increase in March and April and peaked in August. It has remained at this level through early 1995. Data from the provincial veterinary diagnostic laboratories, the number of herds with a positive BVD diagnosis increased from 132 in 1991 and 120 in 1992 to 356 in 1993 and 353 in 1994.

The data from the provincial veterinary laboratories underestimates the actual number of affected herds. A mail survey of veterinarians in Ontario was conducted in the fall of 1993. Forty-seven practices responded that provided service to a total of 3695 dairy herds, 2692 beef herds and 398 veal operations. Of the dairy herds, 112 had a diagnosis of BVD, only 48% of the diagnoses were made through VLS laboratories. Of beef herds, 169 had a BVD diagnosis with 27% of the diagnosis made in a VLS laboratory. Eighty-five veal barns had a BVD diagnosis, with 22% of the herds using VLS for the diagnoses.

As well as an increase in the number of farms with positive BVD diagnoses, there was an increase in the severity of outbreaks. The following cases illustrate the severity of outbreaks.

Case 1:

In June 1993, the owner of a 56 milking cow Holstein dairy herd purchased 11 heifer calves at a sales barn. These calves were placed directly in a calf barn that already contained 36 animals ranging in age from under 1 month to yearling steers. The farmer had not vaccinated.

In the 3 weeks after introducing the calves, all the purchased and resident calves developed respiratory disease and diarrhea. Five died and three were examined at post mortem; 2 had chronic pneumonia and 2 had lesions of BVD/MD. Five of 12 steers in the same barn also became ill with signs of pneumonia and diarrhea/dysentery. Two steers examined at the Kemptville V.L.S. Laboratory had extensive ulceration of the oral mucosa, esophagus and small intestines and histological lesions compatible with BVD/MD. By the sixth week after bringing the purchased calves onto the farm, 20 calves, 5 steers and 4 fresh second calf heifers had died with clinical signs or lesions of BVD/MD. The 4 second calf heifers became ill within a few days of each other. Their illness

represented the first time that BVD had been observed in the milking herd. The owner elected to ship the milking herd to slaughter.

Case 2:

A veterinarian was called to examine 6 calves that had developed respiratory disease. Two calves died. On gross post mortem, the diagnosis was chronic bronchopneumonia but the diagnosis was BVD on histology and virology. Coincidentally, 4 cows were off-feed with pyrexia (41 C) and increased respiratory rate. One of these cows died with mucosal lesions of BVD. Several other cows and calves were seen to be ill over the next few weeks.

Ten cows had been purchased 2 months earlier. These cows were vaccinated on entry but the main herd had never been vaccinated until after the beginning of the outbreak.

Summary of the outbreak:

Age group	Number	Sick	Died
Calves (< 12 mos.)	13	13	12
Heifers	14	8	4
Cows (unvaccinated)	24	14	2 + 7*
Cows (vaccinated)	10	1 (an abortion)	0

*7 cows were salvaged when they first showed signs of the disease.

Overall mortality, 41% (49%, if the purchased cows are excluded).

Case 3:

Between June 8 and July 7, 1993, 20 of 40 heavy (750-800 lbs) Holstein steers died usually within 10-18 hours of developing profuse bloody diarrhea and extensive ulceration in the mouth. At post mortem, ulcers and erosions were also found in the esophagus, abomasum and intestines.

The steers had been assembled from 2 lots purchased from 2 drovers a week before the problem began. They were vaccinated on arrival with an IBR-BVD-PI_3-BRSV vaccine and a Pasteurella bacterin. The group was held in a pen for 3 days then moved to a pasture on June 7. They were fed grain from a wagon on pasture and water was provided by a single water trough. The first steer died on June 8. Subsequently, one or two steers died on June 11, 13, 17, 19, 20, 21, 22, 25, 28 and July 2, 5 and 9. The clinical course of the disease in these animals was extremely rapid. In several cases,

the farm manager reported that cattle appeared to be normal in late afternoon had died by the next morning. Unlike many outbreaks of gastrointestinal disease, in this outbreak there was a slow progression of clinical disease through the group. Usually only one animal was sick at one time. By the conclusion of the outbreak, about equal numbers of steers from each lot had died. Overall mortality was 50%.

Case 4:

Beginning in the second week of September, calves, heifers and cows developed diarrhea. The owner had not vaccinated for several years. The most recent additions occurred in August 1992. A drover had visited the farm a few weeks before the outbreak began. By the second week of October, the outbreak is summarized as:

Age	Number	Dead	Mortality
1-5 mos.	13	6	46%
6-11	13	13	100
12-17	10	3	30
18-23	6	1	17
24-35	17	7	41
36-47	8	1	13
48-128	8	1	13

Overall mortality was 43% before the herd was sold to slaughter.

Case 5:

This is a purebred Holstein herd milking 52 in an older tiestall barn. Replacements are housed in the main barn. Heifers and cows had been vaccinated with a 4-way killed vaccine every year in the spring since 1988. The most recent vaccination had been on April 29, 1993. A primary immunization series had never been administered.

On January 25, 1994, a mature cow became acutely ill with a clinical diagnosis was pneumonia. Two more cows were ill on January 27 and a calf died on February 1. On the next 2 weeks, 6 cows and 6 calves had died. On February 18, the first abortion was seen. The last death from BVD occurred on March 15.

Mortality summary:

Calves and heifers <2 years old:	21/25 (84%)
Lactating cattle	13/52 (25%)
Overall mortality	34/77 (44%)

In the abortion storm, 20 cows and heifers aborted leaving only 4 animals pregnant after the outbreak. All the cattle that were clinically ill subsequently aborted. In addition, abortions occurred in cattle that had not been seen to be sick.

Case 6:

This is a commercial diary herd milking 45 in a new naturally ventilated tie-stall barn. Heifers are reared in a separate barn and at pasture. Dry cows are housed in the tiestall. In the fall of 1991, all the cows in the barn had been vaccinated with a killed 4-way vaccine. A group of 25 heifers at pasture were not vaccinated. Four cows aborted after vaccination. The owner did not vaccinate in 1992.

It is difficult to identify the first animal that became sick with BVD on the farm. A heifer aborted on November 1, 1993 and died 4 days later may have been the first case. A calf died on November 26 and had lesions of BVD on post mortem at the local provincial veterinary laboratory. Two first lactation heifers in adjacent stalls in the tie-stall barn became sick with pyrexia and respiratory signs. They subsequently developed oral ulceration.

On December 1, the twin of the first calf died. During the next week, 10 heifers and another 2 calves died. In second week of December, 4 more heifers died. On December 10, a cow aborted. This marked the first of a series of abortions that continued until February 28. Thirty-two cows and heifers aborted. Only 9 cattle had not aborted; all were mature cows. In total, there were 32 abortions and 16 dead calves and heifers. None of the cows died.

Summary of features of 10 outbreaks:

- 8 tiestall diary farms, 2 beef feeder calf operations
- Range of total number of cattle on farms: 40 to 191, mean 88
- initial clinical complaint: in 6/8 diary herds and 1/2 beef herds, the first complaint was pneumonia in calves or adults; in 2/10 herds, acute diarrhea in older cattle was observed initially; single herds observed abortion or acute BVD/MD as the first cases in the outbreak.

- clinical features: acute BVD/MD (high fever, oral ulcers, diarrhea) was seen in 8/10 herds; in 1/10, abortion was the primary feature; in 1/10, weak calves were the primary feature; in 2/10, sudden deaths (found dead) were prominent.
- range of duration of outbreaks: 4 to 32 weeks
- mortality: mean overall (n=10) 29.6% (range 4.7 to 50%); mean for adults (n=8) 10% (range 0-26%); mean for young stock (n=8) 54% (range 13 - 100%).
- herds experiencing abortions: 7 of 8 (1 herd shipped to slaughter early in outbreak)

number of breeding-age cattle	number of abortions
34	1
52	32
52	20
52	8
70	45
54	4
67	23

- in 9/10 herds, cattle had been purchased in the 4 weeks before outbreaks began.
- in 10/10 herds, cattle were either not vaccinated or not vaccinated according to the vaccine label.

These appear to be outbreaks of acute BVD rather than mucosal disease. This conclusion is based primarily on the pattern of disease occurrence and the high mortality and abortion rates.

What can you do to control this type of outbreak?

Vaccination and isolation of clinical cases may be beneficial in preventing spread of the infection. But there is no certainty that immunity from vaccination will protect cattle during this type of outbreak. Experience in 2 dairy herds in which part of each herd was properly vaccinated and part either not vaccinated or improperly vaccinated indicates that proper vaccination protects against abortion and death although it may not protect against clinical illness. The extremely low number of outbreaks in herds vaccinated according to manufacturers' recommendations also supports recommendations for vaccination.

We also recommend that producers isolate any newly purchased cattle and cattle returning from shows. The quarantine should last at least 3 weeks. Quarantined cattle should not have direct contact with resident cattle and should not share feed bunks, waters and grooming tools. Purchased cattle should be vaccinated and tested for persistent BVD virus infection before entering the herd.

We also advise producers to limiting visitor's access to the barn. Cattle should be transported privately if possible. Trucks and trailers used by commercial carriers should be cleaned before loading new animals.

Should you vaccinate in the face of the outbreak?

It may be beneficial to vaccinate. We do not know the extent that vaccine-induced immunity will protect against acute BVD. Pregnant cows and heifers should be vaccinated to try to prevent fetal infection. We do not recommend the use of modified live virus BVD vaccines in the face of an outbreak.

Which vaccine is best to use?

There is no evidence that one vaccine is better than another. Research in Canada has shown that field isolates from Ontario and Quebec all had some degree of cross reactivity with the vaccine strains of the virus. A large unknown is how cross reactivity in a laboratory translates into protection after natural exposure.

The virology section of V.L.S. in collaboration with the New York State Veterinary Diagnostic Laboratory has investigated the antigenic characteristics of selected BVD viruses from Ontario outbreaks. They determined that these viruses are not antigenically unique and that they share antigens with other known BVD viruses. This information supports the use of currently available vaccines to control BVD in Ontario.

Will isolation of infected cattle prevent spread of the virus?

The BVD virus spreads slowly within a group of cattle apparently because it requires direct or close contact to spread from infected to other susceptible animals. Isolating infected animals or handling them only after uninfected animals have been handled may reduce transmission. The virus is shed in saliva, nasal secretions and other body fluids, so it is recommended that infected cattle should not share water troughs, salt licks and feed bunks with uninfected cattle.

Should you try to identify persistently infected carriers during or after the outbreak?

Persistently infected calves have been born in herds following the outbreak of acute BVD as a result of infection of cows in early gestation. These carriers will maintain the BVD virus in these herds. It is recommended to screen all calves born on the farm for BVD virus once the clinical outbreak has subsided. The most reliable way to identify carriers is by demonstrating the virus usually by isolation from whole blood or serum samples (the type of sample depends on the virology laboratory used). The provincial veterinary diagnostic laboratory uses washed buffy coat cells from newborn calves that have received colostrum. If an animal is positive for BVD virus, it may be a carrier or it may be acutely infected. If it is positive on a second sample collected 3 to 4 weeks after the first then it is likely a carrier. Calves should be kept isolated until test results are known. All carriers should be shipped to slaughter.

Are the outbreaks in Ontario due to a new BVD virus?

The activity of these BVD viruses is different. As mentioned above, information from monoclonal antibody studies shows that viruses from these outbreaks share antigens with other BVD viruses. Many isolates from acute outbreaks in Ontario and Quebec are type 2 BVD viruses. However, 25 to 30% of BVD viruses isolated in Ontario have been type 2 viruses in all but 1 year since 1981. (source: Dr. S. Carman, VLS, Guelph).

Have there been outbreaks in vaccinated herds?

Veterinarians in the Health and Nutrition section of OMAFRA have investigated over 100 BVD outbreaks. Only 1 herd was vaccinated according to manufacturer's recommendations. Many herd owners believed there herds were vaccinated but they had not followed label recommendations.

Outbreaks have occurred in herds that never gave an initial double immunization (primary series) of killed vaccine as directed on the label. Single annual vaccination does not provide immunity to BVD unless preceded by a primary series. There are other situations where vaccination appears to fail. Cattle vaccinated after an outbreak has started or at the same time the virus is introduced into a herd, will be only partially protected. It may protect some animals from getting sick and dying. Unfortunately, vaccination in these situations does not appear to give good protection to pregnant cows and heifers. They may abort or give birth to stunted or weak calves.

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Definitions and descriptions:

Acute BVD is any clinical manifestation of BVD virus infection in a host first infected with BVD virus after birth. The clinical signs of infection range from inapparent infection with seroconversion to severe clinical disease with pyrexia, diarrhea and ulceration of the gastrointestinal tract including Peyer's patches. Thrombocytopenia with hemorrhage ("hemorrhagic syndrome") is one form of acute BVD that has been described in veal calves and adult cattle (Corapi, W.V., French, T.W., Dubovi, E.J., 1989, Severe thrombocytopenia in young calves experimentally infected with noncytopathic bovine viral diarrhea virus. J. Virol. 63: 3934-3943.)

Acute mucosal disease is an acute clinical disease in cattle that were born persistently infected with a noncytopathic biotype of BVD virus. Cattle that die of mucosal disease were first infected with a noncytopathic biotype of BVD virus as a fetus during the first 120 days of gestation. They develop mucosal disease after they subsequently become superinfected with a cytopathic biotype of BVD virus.

Acute mucosal disease is characterized clinically by pyrexia, diarrhea and ulceration of the gastrointestinal tract including Peyer's patches. Acute mucosal disease cannot be differentiated from acute BVD on clinical signs or pathologic lesions.

It is important to distinguish between acute BVD and mucosal disease because the preventive strategies are different for each disease. Acute BVD (and other manifestations of post natal infection) can be controlled by immunization of the cattle at risk. Mucosal disease could only have been prevented by preventing infection of the fetus either by protecting pregnant cows from exposure to BVD virus or by vaccinating the dam before breeding. Vaccination of an animal that is born persistently infected with BVD virus will not protect that animal against developing mucosal disease.

Cytopathogenicity refers to the affect that a BVD virus has on cells used for culture of BVD virus in the laboratory. Cytopathic viruses cause visible damage to a cell monolayer. Noncytopathic BVD viruses do not cause detectable damage. Cytopathogenicity is not an indication of virulence. Eighty-eight percent of BVD viruses isolate in Ontario in 1993 were noncytopathic (source: Dr. S. Carman, VLS, Guelph)

The terms **type 1 and type 2 BVD viruses** refer to a classification of viruses based on the presence of specific nucleic acid sequences in their RNA. Many of the BVD viruses from acute outbreaks in Ontario and Quebec were type 2 BVD viruses. Type 2 BVD viruses are also commonly isolated where there has been is no clinical disease. Type 2 BVD viruses have isolated in Ontario almost every year since at least 1981 (source: Dr. S. Carman, VLS, Guelph).